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Massive body-brain disconnection consequent to spinal cord injuries drives profound changes in higher-order cognitive and emotional functions: A PRISMA scoping review

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ABSTRACT

Spinal cord injury (SCI) leads to a massive disconnection between the brain and the body parts below the lesion level representing a unique opportunity to explore how the body influences a person's mental life. We performed a systematic scoping review of 59 studies on higher-order cognitive and emotional changes after SCI. The results suggest that fluid abilities (e.g. attention, executive functions) and emotional regulation (e.g. emotional reactivity and discrimination) are impaired in people with SCI, with progressive deterioration over time. Although not systematically explored, the factors that are directly (e.g. the severity and level of the lesion) and indirectly associated (e.g. blood pressure, sleeping disorders, medication) with the damage may play a role in these deficits. The inconsistency which was found in the results may derive from the various methods used and the heterogeneity of samples (i.e. the lesion completeness, the time interval since lesion onset). Future studies which are specifically controlled for methods, clinical and socio-cultural dimensions are needed to better understand the role of the body in cognition.

1. Introduction

Traditional theories (e.g. "the sandwich model", Hurley, 2001) posit that an individual's mental life relies on higher-order cognition and emotions and that somatosensory and motor bodily representations are purely instrumental, ancillary components. Only recently, a number of new theoretical approaches, grouped under an umbrella definition entitled Embodied Cognition Theories (ECT), have advanced the idea that all human experience is grounded on sensorimotor processes, and thus on the body (e.g Glenberg, 1997; Gallagher, 2005).

Despite the fact that a great deal of research into ECT has been done in the last twenty years (for review see Jacquey et al., 2019; Ianì et al., 2021; Signorelli et al., 2022; Borghi et al., 2022), the issue of the direction of the body-brain influence (from sensorimotor to symbolic or viceversa) is still up for debate. Is it the body that gives rise to cognitive activity (O'Reagan & Noe, 2001; Barsalou, 2008) or the symbolic system that merely activates motor responses while executing cognitive tasks (Leshinskaya and Caramazza, 2016)? Although advanced techniques (e.g. virtual reality environments and bodily illusion settings) have made it possible to investigate the body-brain relationship in depth (Cerasa et al., 2022; Pyasik et al., 2022; Snow and Culham, 2021 for review), all of the experimental attempts to gain knowledge in this area have been hampered since only partial results can be achieved due to the fact that it is impossible to effectively separate the body from the brain in healthy people.

Interestingly, there are some clinical conditions, such as Spinal cord injuries (SCI) and Locked-in syndrome (LIS), that are specifically characterised by this disconnection. As a consequence of these conditions, the individual suffers body deafferentation (i.e. the interruption of sensory inputs in the pathway towards the brain) and deefferentation (i.e. an interruption to the subcortical motor pathway) although in absence of any direct brain damage. Thus, these conditions represent a natural model for investigating potential cognitive and emotional changes that are mediated by body-related dysfunctions.

Disconnections may involve different portions of the body (i.e. the trunk and/or lower limbs in paraplegia, the upper and lower limbs in tetraplegia, while in Locked-in Syndrome, the paralysis affects facial muscles as well – see Fig. 1 for details). This means that it is possible to

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investigate both the specific changes which are topographically associated with the neurological level of injury (NLI – the most caudal spinal cord segment in which somatosensory and motor functions are fully spared), and also general changes to higher-order cognitive functions.

Previous experimental studies indicate that topographically organised modifications occur after SCI in those cognitive functions that are in some way associated with the body (i.e. corporeal perceptions and illusions, motor imagery, action discrimination and representation and space representation, see Moro et al., 2022 for a review of the literature).

The current study aims to extend the abovementioned analysis in order to investigate cognitive functions and emotional responses that are not necessarily characterised by the topography of the body-brain disconnection, such as attention, memory, executive functions and emotion discrimination. From a neurophysiological point of view, it is possible that these processes are supported by underlying neural plasticity caused by body-brain disconnection. To date, plasticity is considered to be a fundamental mechanism of the nervous system, a continuous process that, throughout an individual's life, builds and rebuilds neural networks depending on the stimuli and requests coming from the environment (Berlucchi and Buchtel, 2009; Berlucchi, 2011). Studies on healthy subjects demonstrate that these processes may be fast and reversible, and may involve not only sensorimotor functions, but also cognition, emotions and social behaviour (Froemke and Young, 2021; Böckler and Singer, 2022; Goto and Hayashi, 2022; Lee et al., 2022; Mudgal et al., 2022; Said et al., 2022).

Furthermore, postlesional plasticity has been extensively documented after damage to the central and peripheral nervous systems (Bashir et al., 2010; Merabet and Pascual-Leone, 2010; Makin and Flor, 2020; Ryan et al., 2021). In individuals with SCI, post-lesional plasticity has been demonstrated to extend beyond the somatosensory and motor networks (Çermik et al., 2006; Castro et al., 2007; Kokotilo et al., 2009; Aguilar et al., 2010; Freund et al., 2011; Henderson et al., 2011; Nardone et al., 2013), and to also involve the associative structures that are implicated in cognitive functions (e.g parietal lobes and cerebellum, Solstrand Dahlberg et al., 2018; Ilvesmäki et al., 2017; Wrigley et al., 2009; Curt et al., 2002) and emotions and motivation (e.g. reward network, Gustin et al., 2010).

Despite this evidence, research into the relationship between these neuroplastic modifications and cognitive and emotional reactions is still meagre, and even more importantly, is not really considered for specific rehabilitation interventions. The aim of the current study is to fill this gap by means of a PRISMA scoping review focused on studies investigating cognitive and emotional changes following body/brain deafferentation and deefferentation.

2. Methods

A pre-registered (https://doi.org/10.17605/OSF.IO/52ZJ8) scoping review (Arksey and O'Malley, 2005) was performed to answer the main research question, that is, whether changes to cognition and emotions occur as consequence of body/brain disconnections without direct brain damage. The review concentrated on acquired neurological damage such as Spinal cord injuries and Locked-in syndrome, while peripheral lesions associated with changes in body shape (e.g. amputations) were not considered.

2.1. Information sources and search strategy

A comprehensive, systematic search of the literature was carried out with reference to PubMed and ScienceDirect for articles published between 1986 and 2023, entering the following keywords: "Deafferentation" AND "Change in Cognition"/ "Cognition; "Somatosensory System" AND "Cognition"/ "Emotion"/"Motor Deprivation in Cognition"; "Amyotrophic Lateral Sclerosis" AND "Cognition"/ "Emotion"; "Spinal Cord Injury" AND Cognition" / "Emotion"; "Locked-in Syndrome" AND "Cognition" / "Emotion"; "Polyneuropathy" AND "Cognition" / "Emotion".

2.2. Study selection and data extraction

After removing duplicates and non-English literature, those which were eligible then went through a first selection based on title and then on abstract scanning, followed by a third full-text assessment phase. All of these steps were conducted independently by two of the authors (VM and MB). In cases where there was disagreement, the third author (MS) or the fourth (SMA) were consulted for the final decision. The search and selection of studies followed the PRISMA2020 guidelines (Page et al., 2021), as illustrated in the flow-chart below (Fig. 2). Studies retrieved from the websites of associations for individuals with SCI (n = 6) and organisations (https://asia-spinalinjury.org/, https://www.sci-info-pages.com/spinal-cord-injury-organizations/, https://www.pro-bed.com/blog/info/top-10-spinal-cord-research-organizations,

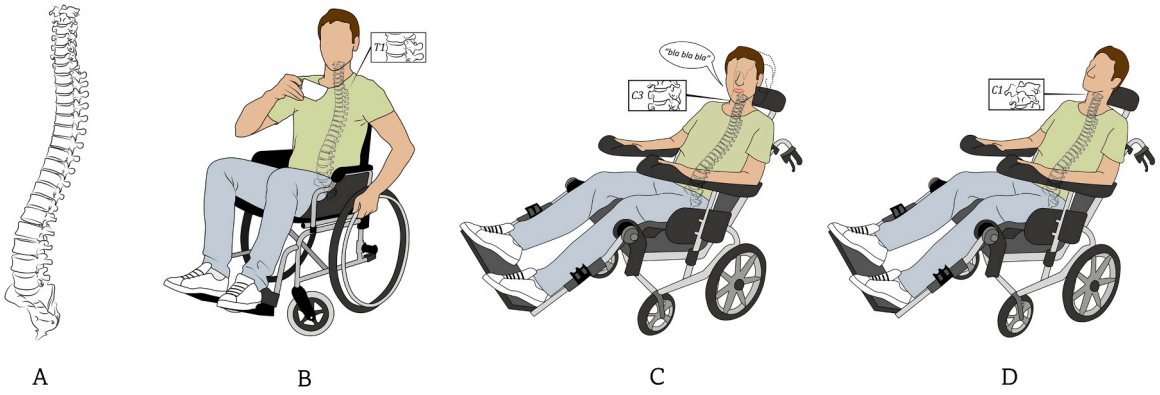

Fig. 1. Sensorimotor deficits following Spinal cord injury (SCI) and Locked-in Syndrome (LIS). A) A schematic representation of the spine; B) Lesions below the first thoracic level of the spinal cord (T1) cause paraplegia, with sensory and motor impairments involving the chest, the trunk, and the lower limbs (according to the neurological level of injury). Sensory and motor functions in the upper limbs and the head are spared; C) The effects of lesions that occur at or above T1 (C3, in the figure) with tetraplegia, a condition leading to sensory and motor impairments that include upper limbs and the muscles necessary for breathing (i.e. the diaphragm muscle), with neck and head spared; D)Ventral brain stem lesions, with whole body paralysis (but not sensory deficits) associated with LIS. SCI lesions can be complete (with no function and global anesthesia in the body parts innerved by the spinal segments at the NLI and below) or incomplete (with varying degrees of residual motor activity and sensory impairment). The clinical index of lesion completeness is the absence of sensory and motor functions in the peri-anal area, which is innerved by the S3–4 spinal segments. LIS is associated with anterior lesions in the brain stem and midbrain resulting in motor deficits in the whole body but spared sensory functions.

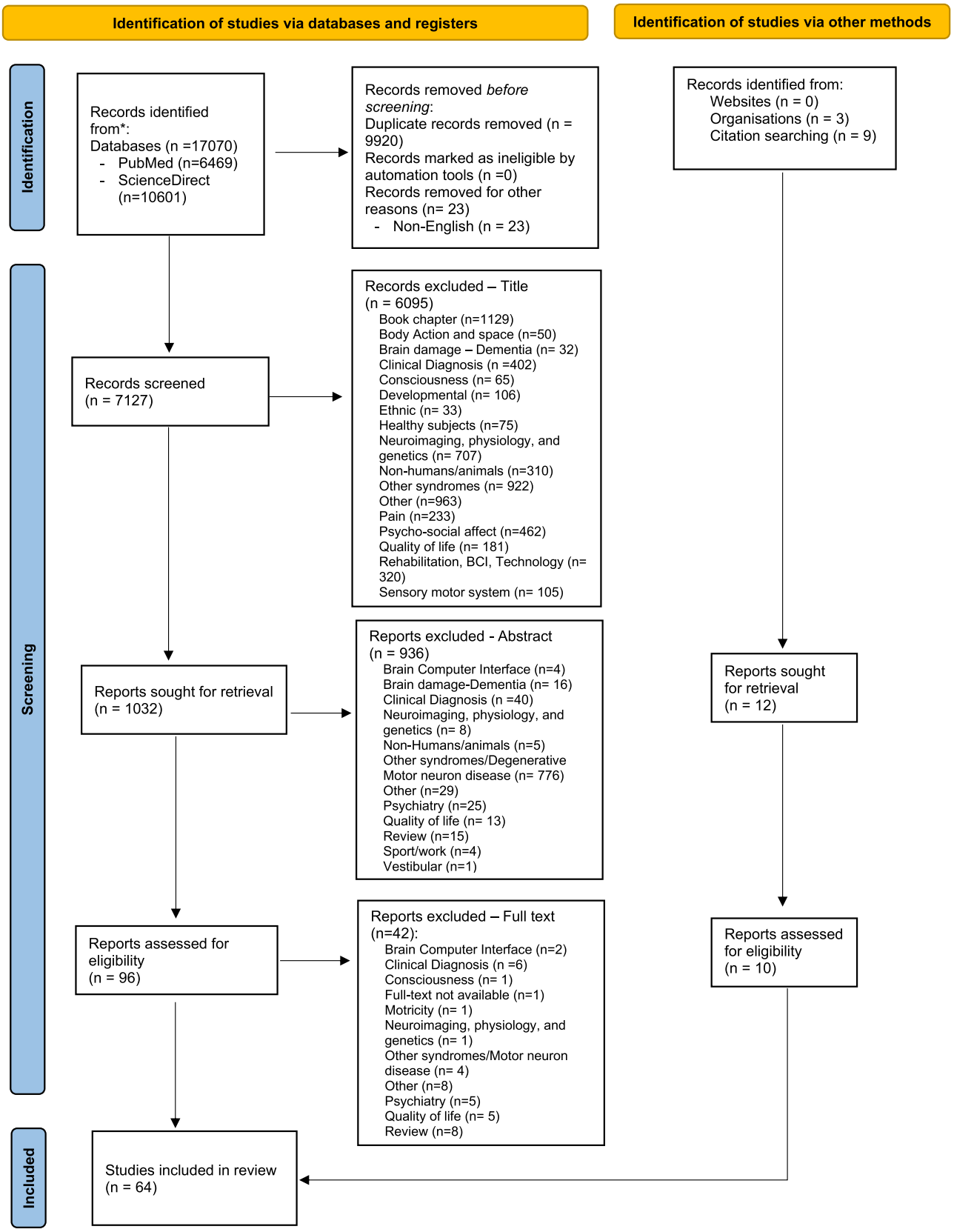

Fig. 2. PRISMA Flow diagram illustrating the steps followed in the literature search and article screening.

n = 14) were included and cross-references were also checked.

Another search which focused on neuropathies and other peripheral syndromes did not yield relevant results for the topic in question (see SM-1).

3. Results

3.1. Study selection

The initial search identified 17,070 potentially relevant titles (6469 in Pubmed and 10,601in Science Direct). After removing the duplicates, 7127 articles remained. A total of 1032 articles survived the title scanning phase conducted according to the selection criteria devised (see Fig. 2 for details). Then, a further selection was performed based on the abstracts, and the remaining 96 articles underwent the full-text assessment phase. During the scan of the abstracts, studies on Amyotrophic Lateral Sclerosis were excluded ("Degenerative motor neuron diseases" in the PRISMA) since direct brain damage occurs in the very early stages of the illness and deterioration in cognitive functions is reported as a symptom of the disease making it difficult to disambiguate any changes associated with sensory and motor deficits due to cerebral damage (e.g. Amato et al., 2012; Uher et al., 2014; Petracca et al., 2021).

Based on the full-text assessment, 54 articles met the selection criteria. Moreover, 12 other additional articles were identified via other sources, 9 from citation searching and 3 from a consultation of the websites of various relevant organisations. Among these, 2 papers were excluded since they were duplicates, while the remaining 10 articles were included in the selection. The final number of records which were eligible came to 64, 43 of which mainly considered cognitive functions and 21 which focused on emotional processes.

3.2. Characteristics of the studies which mainly considered cognitive functions

The studies on cognitive changes following SCI and LIS are summarised in Table 1. Among the 43 studies on the cognitive domain, 10 investigated multiple different cognitive functions by means of general neuropsychological batteries, while 33 referred specifically to one or more specific functions. The articles constituting this latter group were classified according to the cognitive domains which they investigated (25 on attention and executive functions, 19 on memory and 8 on language) and these are subsequently discussed in separate paragraphs. Furthermore, two specific sections of the results are dedicated to the effects of autonomic functions and drugs on cognition.

3.2.1. General assessments of cognitive functions in individuals with SCI

Results from studies administering neuropsychological batteries for the general assessment of cognitive functions after SCI are inconclusive, both in terms of the incidence of disturbances and co-occurrent variables contributing to an impairment in cognitive performance. Indeed, there is a great deal of variability in the prevalence of cognitive deficits, ranging from 16.6% (Craig et al., 2017) to 60% (Dowler et al., 1997), with even higher rates when the concomitant presence of traumatic brain injury is considered (TBI, e.g. 74.2% in Tolonen et al., 2007). Despite the fact that the majority of the studies included in this review (28 out of 37 studies where the data is reported) considered the co-occurrence of TBI as an exclusion criterion for the recruitment of participants (Table 1), deficits were observed. Furthermore, in a study testing a relatively large sample (117 participants, subacute/chronic phase, interval lesion-test $\geq$ 6 months), the performance of the patients with SCI alone did not differ from those of patients with TBI in comorbidity with SCI. Crucially, in both groups, the scores were lower than the normative scores for all of the neuropsychological tests carried out (i.e. attention, memory and perceptual reasoning, Macciocchi et al., 2013, see also Hess et al., 2003).

The interval between the assessment and the onset of injury was also highlighted as a factor influencing cognitive performance, but again the results are ambiguous. While some studies report that the severity of mild cognitive impairment worsens over time, akin to an accelerated cognitive ageing process (Molina et al., 2018), others indicate that this is true only for patients with low cognitive functioning across multiple domains during the acute phase (Pasipanodya et al., 2021). A recent study (Molina-Gallego et al., 2021) compares two groups of people with acute and chronic SCI ~~people~~, matched for demographic and clinical variables (in terms of NLI and completeness of lesion) as well as for tracts relating to emotions. In ad-hoc chosen, motor free tests (i.e. without motor requirement), the participants with chronic SCI showed impaired performance in attention, processing speed and visual memory and a worsening tendency in terms of learning and recognition (see also Roth et al., 1989; Dowler et al., 1995).

Motor free tests have also been used to assess cognitive functions in patients with Locked-in Syndrome (Schnakers et al., 2008). These revealed the absence of extensive deficits, that is, difficulties emerged only in the final part of the tasks, suggesting incoming fatigue. When specific difficulties in any of these tests were found, the lesions were not confined to the brainstem but also extended to the brain (i.e. thalamic or cortical structural brain lesions) (Kumral et al., 2022). Further evidence of spared cognitive functions in LIS, despite the long-standing deefferentation characterising the condition, comes from two single case reports, in which extensive neuropsychological testing was carried out by means of a communication aid system (writing apparatus) which exploited the patients' residual eye movements (Allain et al., 1998). However, considering the patients' difficulties in giving verbal responses, these tests were based on multiple-choice rather than open responses (Kumral et al., 2022), which makes the tasks potentially easier than standard validated tests.

Other factors are reported to impact on cognitive disorders in individuals with SCI, such as age (Tun et al., 1997), a low standard of education, a history of smoking or substance/alcohol abuse, mood disorders, fatigue (Sachdeva et al., 2018; Pasipanodya et al., 2021), a polypharmacy assumption regime (e.g. antimuscarinics, Krebs et al., 2018), and disorders relating to autonomic/cardiovascular control (Wecht et al., 2016; Wecht et al., 2018; Carlozzi et al., 2022). Moreover, apnoea and sleep disorders (which are common after SCI) were found to be associated with oxygen desaturation and cognitive impairment, in particular in attention, executive functions and working memory (Sajkov et al., 1998; Schembri et al., 2017; Carlozzi et al., 2022, see paragraphs 3.2.4 and 3.2.5).

An unexpected result regards the neurological level of injury (NLI). Indeed, a recent review (Sachdeva et al., 2018) indicates that when considering the relation between the severity of cognitive impairment and the NLI, only 2 out of 14 studies report a correlative effect between NLI and cognition. This data is in contrast with experimental evidence showing the effects of NLI on various body-related cognitive functions (e.g. motor imagery, Scandola et al., 2017a; Vuckovic et al., 2015 and body representation, Pernigo et al., 2012; Scandola et al., 2014, 2017b; Pozeg et al., 2017; Ionta et al., 2016; Fiori et al., 2014).

These discrepancies may depend, at least in part, on the domains tested. In particular, some studies suggest that while the abilities associated with crystallised cognition (i.e. with premorbid functioning, e.g. the Vocabulary test and Oral Reading recognition) do not change as a consequence of a SCI, fluid cognition (i.e. processing speed, visuospatial episodic memory, cognitive flexibility, response inhibition and working memory) is more impaired in tetraplegic than in paraplegic patients, and that this difference persists even after controlling for fine motor speed and dexterity (Cohen et al., 2017; Carlozzi et al., 2017).

Crucially, cognitive impairment influences the psychological well-being of individuals with SCI (Pasipanodya et al., 2021; Houldsworth et al., 2023) and contributes to the development of negative mood states, especially during the transition from the hospital into the community when personal resources are severely challenged (Craig et al., 2017). For these reasons, and considering the potential risk of mild, undiagnosed cognitive difficulties (Cohen et al., 2017; Molina-Gallego

Table 1

Studies on the cognitive changes investigated in the articles resulting from the review of the literature. Data are reported when the performance of the SCI individuals was compared with normative data or control groups. n. = the number of the SCI participants; interval = interval from lesion onset (as this is reported in the original articles); damage severity = completeness of lesion (AIS scale); T = tetraplegics (followed by a percentage when the sample is mixed); w. = weeks; m. = months; y. = years; chronic and subacute are used when no other temporal information is given in the original articles; AIS = American Spinal Injury Association Impairment Scale (Ditunno et al., 1994); A= complete lesion (no somatosensory or motor functions below the neurological level of injury - NLI); B = spared sensory functions below the NLI; C = spared sensory and motor functions below the NLI, but no motricity against gravity; D = spared sensory and motor functions below the NLI, with possibility of motion against gravity; E = preserved sensory and motor functions), otherwise reporting LIS = Locked-In Syndrome. In the third column, (controls), the presence of control groups: no = no control sample; HC = healthy controls; TBI = traumatic brain injury; VS = vascular stroke patients. TBI = co-occurrence of Traumatic Brain Injury in the clinical sample (no= TBI is an exclusion criterion; nr = not reported). GEN NPSY ASS= general neuropsychological assessment; ATT = attention; PROC SPEED = processing speed; EX FUN = executive functions; DEC MAK = decision making; REAS = reasoning; VISUO-CONSTR FUN = visuo-constructional functions; LANG = language; WORK MEM = working memory; VERB MEM = verbal memory; VISUO-SPAT MEM = visuo-spatial memory; FREE MOT SPEED TEST = free motor speed test; ANX = anxiety; DEP = depression; + = presence of deficits; - = no deficits; blank space = unreported.

	n. – interval – damage severity	controls	TBI	GEN NPSY ASS	ATT	PROC SPEED	EX FUN	DEC MAK	REAS	VISUO-CONSTR FUN	LAN GUAGE	WORK MEM	VERB MEM	VISUO-SPAT MEM	FREE MOT SPEED TEST	ANX	DEP
Houldsworth et al. (2023)	41 (T = 7.32%) ASIA: A, B, C,D, E > 6 m (from 7 to 696)	no	no	+			+			+	+		+	+			
Dudley-Javoroski et al. (2022)	23 (T = 47.83) ASIA: A; > 6 m. (from 8 m. to 30 y)	20	no		+		+					+		+			
Carlozzi et al. (2022)	167 (T = 43.11) > 6 m.	no	2.99%		+	+	+				+	+	+				
Kumral et al. (2022)	100 LIS (acute and 1 y follow-up)	no	nr		+												
Goraczko et al. (2022)	9 (T = 44.44) > 4 m.	no	22.22%				+				+	+	+				
Pasypanodia et al. (2021)	89 (T = 65.2%) - 6 m. - AIS A, B, C, D, E	no	30.3%	+	+		+			+	+		+				
Molina-Gallego et al. (2021)	100–50 from 4 to 6 m., 50 > 1 y.	no	no	+	+	+	+						+	+		+	+
Lee et al. (2021)	22 - from 8 m. to 32 y.	HC	no				-					+			+		
Gao et al. (2020)	11 - > 9 m. - AIS A	13	no		-		+						+				
Heled et al. (2020)	20 (T = 10%) - > 10 m. - AIS A, B, C	HC	no		+		+				-		+				+
Chiaravallotti et al. (2020)	59 (T = 50%)	HC	no		-	+	+						+				
Lucci et al. (2019)	18 (lesion between T4 and L1) – from 1 to 9 m AIS A, B, C	HC	no		+												
Molina et al. (2018)	66–32 from 4 to 6 m., 34 < 1 y.	HC	no		+	+	+						+	+		-	+
Maresca et al. (2018)	1	no	nr	+	+		+		+	+		+	+			+	+
Krebs et al. (2018)	29 (T = 34.48%) -AIS A-C	no	no		-		-			-			-				
Wecht et al. (2018)	67 (T = 50%)	HC	no				+										
Handrakis et al. (2017)	8 (T) - chronic - AIS A, B	HC	no		+		+					+					
Craig et al. (2017)	150 (T = 58%) - chronic - 47% = AIS A	HC	no	+	+		+			+	+		+			+	+
Cohen et al. (2017)	156 (T = 41%) - 12 m. – AIS A, B, C, D, E	HC	nr	+	+	+	+				-	+		+			
Schembri et al. (2017)	104 (T + breathing disorders) - < 3 m.	no	no		+	+	+		+		-		-				
Srisim et al. (2017)	13 - ≥ 53 m. - AIS B, C, D, E	HC	nr		+		+										
Carlozzi et al. (2017)	188 (T = 51%) - ≥ 12-m.	TBI and VS	no	+	+	+	+				-	+		+	-		

(*continued on next page*)

Table 1 (*continued*)

	n. – interval – damage severity	controls	TBI	GEN NPSY ASS	ATT	PROC SPEED	EX FUN	DEC MAK	REAS	VISUO-CONSTR FUN	LAN GUAGE	WORK MEM	VERB MEM	VISUO-SPAT MEM	FREE MOT SPEED TEST	ANX	DEP
Wecht et al. (2016)	15 (T = 80%) -chronic - AIS A, B, C	HC	nr				+										
Allison et al. (2017)	20 (T = 55%) - > 2 y. - AIS A, B, C, D	no	nr										+				
Phillips et al. (2017)	35 (lesion at or above t4) - > 10 y.	HC	no				-										
Handrakis et al. (2015)	7 (T) – chronic - AIS A, B, C	HC	no		-		-						-				
Phillips et al. (2014)	10 >T6 - > 1 y. - AIS A, B	HC	1				+										
Macciocchi et al. (2013)	117 - < 3 m.	no	53		+	+			+			+		+			
Lazzaro et al. (2013)	37 (T = 27%) - chronic -AIS A, B, C, D, E	HC	no		+										+		
Wecht et al. (2012)	13 (T = 53.85%)	HC	no				+										
Jejede et al. (2010)	20 (T = 65%) - from 2 to 29 y.	no	45%		+	+	+				-		+				
Schnakers et al. (2008)	10 LIS - 9 chronic, 1 subacute	HC	1		+				+		+		+		+		
Tolonen et al. (2007)	31 (T = 51.61%) - 5 m - AIS A, B, C, D	no	74%	+	+		+		+				+	+			+
Hess et al. (2003)	33 (T = 40%) - 1 m.	TBI	no		+	+	+			+		+	+				
North and O'Carroll (2001)	20 (T) - > 6 m.	HC	no	-	-		-	-			-		-			-	-
Sajkov et al. (1998)	37 T (11 +sleep disordered breathing) -> 6 m. - AIS A, B, C	no	no		+		+			-		+					-
Allain et al. (1998)	2 LIS - 2–3 y.	no	no						+		+		+		+		
Tun and Tun (1997)	46 (T = 28.26%) - > 12 m - Young vs. elderly	no	no		+							+	+				+
Dowler et al. (1997)	91 - > 12 m.	HC	yes		+	+	+						+	-	+		+
Cohen et al. (1996)	30 (T = 50%)	HC			+												+
Dowler et al. (1995)	75 (T = 12.3%) - > 1 y	HC	no		-	+	-			-	-	-	-	-	+		
Cohen et al. (1992)	16 (T = 81,25)	HC	no		+												+
Roth et al. (1989)	81	HC	no	+	+		+				+		+		+		

Table 2

Studies on the emotional changes investigated in the articles resulting from the review of the literature. n. = the number of SCI participants; interval = interval from lesion onset (as this is reported in the original articles); damage severity = completeness of lesion (AIS scale); T = tetraplegics (followed by a percentage when the sample is mixed); w. =weeks; m. = months; y. = years; chronic and subacute are used when no other temporal information is given in the original articles; AIS = American Spinal Injury Association Impairment Scale (Ditunno et al., 1994); A= complete lesion (no somatosensory or motor functions below the neurological level of injury - NLI); B= spared sensory functions below the NLI; C = spared sensory and motor functions below the NLI, but no motricity against gravity; D = spared sensory and motor functions below the NLI, with possibility of motion against gravity; E = preserved sensory and motor functions), otherwise reporting LIS = Locked-In Syndrome. the third column, (controls), the presence of control groups: no = no control sample; nr. = not reported; HC = healthy controls; Orthop. = orthopedic patients; TBI = co-occurrence of Traumatic Brain Injury in the clinical sample (no= TBI is an exclusion criterion; yes = co-occurrence of TBI); COGN ASS. = assessment of cognitive functions; EMOT Q. = Emotional quotient; BODY RELATED = Body-related emotions; EMOT. FACES = experimental tasks on emotional face recognition; EMOT. SCENES = experimental tasks on the discrimination of pictures/scenes evoking emotions; EMOT. LEARNING = emotional learning; ANX = anxiety; DEP = depression; INTENSITY REPORTED = reported changes in emotional intensity; nr = not reported; + = presence of deficits; - = no deficits.

	n. – interval – damage severity	Controls	TBI	Methodology	COGN ASS	EMOT Q	BODY RELATED	EMOT FACES	EMOT SCENES	EMPATHY	EMOT LEARN	ANX	DEP	ALEXITHYMIA	INTENSITY REPORTED
Buchtler et al. (2021)	8 (T = 25%) - 3–5 m.	no	yes	Interviews	no		+					+	+		
Guadagni et al. (2019)	20 - AIS B, C	HC	nr	Questionnaires and Exp Tasks	no				-	-		-	-	-	
Eaton et al. (2018)	371 (T = 48.2%) - AIS A, B, C, D	no	nr	Questionnaires	no							+	+		
Lim et al. (2017)	3556 - AIS A, B, C, D, E	HC	nr	ICD-9-CM based	no							+	+		
Saberi and Ghajarzadeh (2017)	110 (T = 18,3%)	HC	nr	Questionnaire	no	+				-					
Kennedy and Hasson (2016)	44 (T = 45.4) a subgroup from Kennedy et al. (2000) - > 16 y	no	nr	Questionnaires	no								+		
Pistoia et al. (2015)	10 (T) - (<1 y) - AIS A	HC	no	Exp Tasks	yes			-	+						
Willliams et al. (2014)	204 - acute, subacute and chronic < 50 y. - AIS A, B, C, D, E	no	nr	Questionnaires	no								+		
Saunders et al. (2012)	801 (T = 39.8%) - > 2 y. - AIS A, B, C, D, E	no	nr	Questionnaire	no								+		
Deady et al. (2010)	24 (T)	HC – Orthop.	no	Exp Tasks	yes						-		-	-	
Pistoia et al. (2010) (LIS)	7 - > 6 m.	HC	yes	Exp Tasks	yes			+	-						
Nicotra et al. (2006)	7 (T = 2) - > 14 w. -AIS A, B, C, D,	HC	nr	Questionnaires and Exp Tasks - Autonomic responses and fMRI	no						+	-	-		
Dryden et al. (2005)	201 (T = 53.7%) - < 6 y. - AIS A, B, C, D, E	no	yes	Questionnaire	no								+		
O'Carroll et al. (2003)	20 (T) - > 2 y. -AIS A	HC	no	Questionnaires	no							-	-	-	
Cobos et al. (2002)	19 (T = 21,05%) -> 18 m.- AIS A, B, C, D	HC	nr	Exp Tasks and Autonomic responses	no				-						
Kennedy and Rogers (2000)	104 (T = 44%) - < 2 y.	no	nr	Questionnaires	no							+	+		

(*continued on next page*)

Table 2 (*continued*)

	n. – interval – damage severity	Controls	TBI	Methodology	COGN ASS	EMOT Q	BODY RELATED	EMOT FACES	EMOT SCENES	EMPATHY	EMOT LEARN	ANX	DEP	ALEXITHYMIA	INTENSITY REPORTED
Kennedy et al. (2000)	87 (T = 32%) - < 2 y. - AIS A, B, C, D, E	no	nr	Questionnaires								+	+		
Montoya and Schandry (1994)	13 (T = 46.15%) -> 14 m. - AIS A	HC	nr	Questionnaire Heartbeat recording	no							-			reduction
Bermond et al. (1991)	37 - ≤ 9 y. - AIS A, B, C, D	no	nr	Structured interviews and Questionnaires	no										increase
Davidoff et al. (1990)	66, - 2 m.	HC	yes	Questionnaires	yes								+		
Chwalisz et al. (1988)	18 - > 3 y.	HC/ neurological patients	nr	Interviews and Questionnaires	no										increase

et al., 2021; Sandalic, 2022), there is agreement on the need to administer neurocognitive screening in people suffering from SCI, especially during intensive rehabilitation (Craig et al., 2017; Pasipanodya et al., 2021; Sandalic et al., 2022) when cognitive strategies and ad-hoc cognitive- motor integrated training may be implemented (Maresca et al., 2018; Scandola et al., 2019a; Moro et al., 2021). However, recent research addressing more specifically the relationship between cognitive functioning and quality of life in SCI led to inconclusive results (Houldsworth et al., 2023; Dudley-Javoroski et al., 2022; Goraczko et al., 2022).

3.2.2. *Attention and executive functions*

Regardless the concomitant presence of TBI, the most frequently impaired functions after SCIs are attention and executive functions (36 studies out of 41 where these functions are investigated, Table 1). Reduced selective attention and processing speed have been reported in both experimental (e.g. Pernigo et al., 2012; Ionta et al., 2016; Scandola et al., 2019b; Lucci et al., 2019) and clinical investigations (e.g. Molina et al., 2018; Molina-Gallego et al., 2021). Furthermore, electrophysiological investigations show the attenuation of event related potentials (ERPs) in response to stimuli presented in various sensory modalities (Cohen et al., 1992, 1996). In a first study (Cohen et al., 1992), the intensity of auditory and visual stimuli was manipulated, and the effect on ERPs was measured at C3 and C4. When compared to the healthy controls, both the paraplegic and tetraplegic groups showed attenuated cortical responses, with flatter ERPs in tetraplegic patients. Since the difference was more evident in the Nl00-P200 components, the results were interpreted as reflecting the processing of the physical properties of the stimuli as well as selective attention processes. Similar results were found in another study by the same group which addressed the somatosensory domain by means of a tactile oddball paradigm, and a recording of the P300 (Cohen et al., 1996). Transcutaneous electrical stimulation was administered to the neck (i.e. an area where somatosensory functions are spared in both paraplegics and tetraplegics) and the participants were instructed to close their eyes and count the number of stimuli perceived on the target side while ignoring all of the others which were being applied to the other side (i.e. 25% on right -odd- and 75% on the left, or vice versa). The SCI groups exhibited attenuated P300s for all of the sites as compared to the control group, although only the tetraplegic participants were significantly different from the controls. However, this difference was found for both target and non-target stimuli, suggesting non-specific effects. More recently, a pattern of intrusive/impulsive processing, characterised by greater attentional focus allocated towards task irrelevant stimuli (i.e. non-target events) has been reported in an auditory oddball task where participants with SCI showed a significantly increased number of intrusive errors and attenuated P300 in both left and right posterior regions (Lazzaro et al., 2013).

Taken as a whole, these data suggest the possibility that attentional disorders may occur in individuals with SCI at the earlier phases of stimulus detection, with potential interference to subsequent cognitive processing. However, although promising, these data need to be replicated and controlled for clinical variables (e.g. NLI and completeness of lesion and the interval from lesion onset), as well as other concomitant factors. For example, a role relating to mood and emotional reactivity may impact performance during attentional tasks (Maresca et al., 2018; Heled et al., 2020), and very few data are available regarding the impact of other co-occurrent factors (see paragraphs 3.2.4 - 3.2.5).

In more complex tasks (e.g. dual tasks, inhibitory control, attentional shift and reaction times-based scoring), motor responses are usually requested and may artificially reduce performance even when there is no upper limb paralysis but only a reduced control of arm and trunk movements (Lee et al., 2021; Lucci et al., 2019). In-depth investigations of potential deficits in dual tasks (i.e. tasks that require participants to divide cognitive resources in order to execute two things simultaneously), inhibitory control (i.e. the capacity to inhibit an automatic

response to give a less immediate but correct response) and attentional shifting (i.e. the capacity to shift the attentional focus from one target to another) are crucial to rehabilitation programmes after SCI (Gao et al., 2020). Indeed, the impact of these disorders has been found to increase when SCI patients are involved in the recovery of gait and postural control in the presence of incomplete lesions. Although specific neuropsychological measures during these motor tasks are currently lacking, there is evidence indicating that, in these situations, the incorporation of cognitive tasks interferes with motor performance (Costa et al., 2016; Tse et al., 2017; Srisim et al., 2017; Amatachaya et al., 2019) with potential consequences in terms of reduced autonomy and the risk of falls.

3.2.3. Language and memory

In the comprehensive neuropsychological assessments considered in the present study (e.g. Molina et al., 2018; Heled et al., 2020; Molina--Gallego et al., 2021, Table 1), no relevant language deficits, either in comprehension or in oral production, were found following SCI. When we controlled for TBI co-occurrence (i.e. when TBI is considered as an exclusion criterion during recruitment), significant differences between the controls and both paraplegics and tetraplegics were found only in verbal fluency, in particular phonemic fluency (i.e. a task requiring participants to list as many words as possible based on an initial letter, Chiaravalloti et al., 2019) that, however, is mainly considered an index of executive functions (and also for neuroanatomical correlates, Biesbroek et al., 2021).

The results regarding memory and learning differ. People with SCI exhibit a reduced performance in both short-term (Heled et al., 2020; Gao et al., 2020; Molina-Gallego et al., 2021) and long-term memory (Allison et al., 2017; Heled et al., 2020; Gao et al., 2020; Molina-Gallego et al., 2021), as well as in working memory (Lee et al., 2021) and episodic memory, even when motor components are ruled out and only accuracy (rather than response speed) is considered (Lee et al., 2021). Moreover, when the lesion level is taken into account, no differences associated with this clinical variable have been found between patients with paraplegia and tetraplegia (Chiaravalloti et al., 2019).

Visuo-spatial memory represents a largely unexplored field, although 6 out of the 8 studies that address this function report deficits in individuals with SCI (Table 1). This finding is consistent with experimental studies showing changes in space perception and representation following SCIs, in both peripersonal space (i.e. the space around the body, Scandola et al., 2020) and extra-personal space (i.e. the space far from the body, Scandola et al., 2019b) and in the perception of one's own body in space (Scandola et al., 2017b), with consequent limitations in terms of the patients' autonomy in daily life. For this reason, visuo-spatial memory and learning deserve specific attention and further in-depth investigations.

Finally, emotional memory was assessed in an experimental study by means of an ad-hoc devised task (Deady et al., 2010, see Table 2) in which participants listened to a short, emotionally arousing story accompanied by the presentation of some slides. They then did a free recall test and a recognition memory test, and the number of slides correctly recalled was calculated. Although in the verbal memory task the performance of the SCI participants was worse than that of the controls, no differences emerged in the emotional memory domain (Deady et al., 2010).

3.2.4. Bodily-autonomic functions and cognition in individuals with SCI

Although it is well known that individuals with SCI, in particular those with cervical lesions, often present with autonomic dysfunctions (e.g. in thermoregulation, blood pressure or breathing) as a consequence of the interruption of sensorimotor and autonomic pathways (Thayer et al., 2016), the number of studies specifically investigating the effects of these disorders on cognition are meagre. However, the main results converge on the idea that autonomic system alterations impact on cognitive functions, in particular attention (as tested by the inhibitory control task, Phillips et al., 2016), working memory and executive functions (Handrakis et al., 2015, 2017).

Chronic hypotension in individuals with SCI has been found to be associated with deficits in memory, attention and processing speed also when demographic and education parameters are taken into account (Jegede et al., 2010; Wecht et al., 2018; Wecht et al., 2012). Hypothermia and thermoregulation disorders also impact on cognitive functioning (Handrakis et al., 2015, 2017) and the manipulation of body temperature modulates cognition. Inducing normothermia by means of heat exposure (i.e. acclimation from a baseline of 27–35° for up to 120 min) has been found to ameliorate performance in attention, working memory and the speed of task execution in SCI individuals but not in healthy controls (Handrakis et al., 2017).

Finally, an investigation of the effects of breathing disorders and sleep apnoea revealed that nocturnal desaturation (but not the frequency of apnoea and sleep arousals) is associated with lower performance in verbal attention and concentration, immediate and short-term memory, cognitive flexibility, internal scanning and working memory (Sajkov et al., 1998). In a multicentric international study (involving 11 different sites and 104 participants), patients in the post-acute stage (i.e. 2 months post-injury) suffering from tetraplegia and sleep breathing disorders were studied via polysomnography and neuropsychological tests (Schembri et al., 2017). The results confirmed that sleep disorders are associated with poorer attention, information processing and immediate recall but not with long-term memory. More recently, a study by Carlozzi and collaborators (2022) investigated the relationship between sleep and cognitive functioning in a large sample of SCI patients (167) using both objective and subjective measures. The authors found that while subjective ratings of sleep were not related to either objective or self-reported measures of cognitive functioning, there were some relationships between objective measures of sleep (i.e. autonomic nervous system activity recorded during sleep) and objective cognitive performance: lower blood volume pulse and higher electrodermal activity were associated with poorer performance on tasks measuring executive functions and memory.

Although further data are required to better understand the potential implications of these aspects for the acquisition of functions and skills during rehabilitation and the recovery of autonomy in daily life, these results underline the importance of considering these bodily aspects, not only for clinical purposes but also for a better comprehension of the mechanisms and processes underlying human cognition (Moro et al., 2022).

3.2.5. The effects of drugs on cognition in individuals with SCI

Cognitive deterioration and fatigue in SCI people undergoing chronic pharmacological therapies represents a concern for both clinicians and patients (Krebs et al., 2018; Kessler et al., 2011; Lee et al., 2010), although our research indicates that only a few studies have addressed the topic. Contrasting results have been reported for midodrine hydrochloride, an agent used to mitigate blood pressure falls due to adrenergic impairment.

Indeed, while this seems to improve verbal fluency (Phillips et al., 2014), no effects of this drug were found on other executive functions (i.e. two serial subtraction tasks) in a study in which hypotensive SCI patients were compared to a group of normotensive SCI people (Wecht et al., 2016). Indeed, although in the post-drug condition (after the assumption of midodrine hydrochloride and nitro-L-arginine methyl ester) the mean arterial pressure and the mean cerebral blood flow velocity increased during cognitive activation in hypotensive subjects with SCI, no effects on cognitive performance were recorded in any of the groups (Wecht et al., 2016).

The deleterious effect of antimuscarinics (a category of drugs widely used for neurogenic lower urinary tract dysfunction) on cognition has been also investigated in individuals with SCI (Krebs et al., 2018) during the first three months of treatment. Although cognitive deterioration was not recorded, the authors conclude that only longitudinal studies with larger, age-stratified samples will lessen the concerns regarding the

effects of antimuscarinics on cognition. No studies on the correlations between anti-spasticity drugs (e.g. Bacoflen, Tizanidine and Dantrolene) and cognitive functions in individuals with SCI were found in our research of the literature, although there is agreement on the necessity to investigate the effects of confounding factors such as anti-spasticity medication on cognitive performance (Rupp, 2020; Holtz et al., 2018).

Another mechanism which is considered to contribute to cognitive decline is chronic neuroinflammation (Vints et al., 2023). The effects of an anti-inflammatory diet on cognitive functions (memory and verbal learning) have also been investigated by Allison et al. (2017). In this study, inflammatory markers (pro- and anti-inflammatory cytokines, tryptophan, kynurenine and several large neutral amino acids) were measured in the serum during three testing sessions (baseline, 1 and 3 months) in an experimental group and in a control group (i.e. with no intervention). The participants were asked to eliminate foods with high glycemic indices (e.g. refined wheat products and refined sugars), foods subject to common intolerances (e.g. cow's milk) and foods that negatively influence cardiovascular health (e.g. hydrogenated oils). Omega 3, Chlorella, antioxidants, curcumin and a vegetable-based protein powder were consumed as daily supplements. Despite the resulting reduction in inflammation markers, no significant effect was found in the experimental group in any of the memory and learning measures considered (i.e. short-term recall, learning slope, long delay free recall, intrusions and repetitions). However, since the participants were at a chronic stage of their illness (i.e. with years of previous chronic inflammation), the authors suggest that it is possible that the underlying hippocampal damage (Jure and Labombarda, 2017) may have prevented potential cognitive improvements or that the reduction in inflammation achieved was insufficient to induce substantial changes in indices of verbal learning and memory. Longitudinal studies, associated with neurophysiological and neuroanatomical measures, are necessary.

3.3. Studies which mainly focus on emotions in individuals with SCI

21 studies mainly focusing on emotional changes after SCI respected the selection criteria. These are summarised in Table 2. The majority of the studies eligible refer to depression (n. 14) and anxiety (n. 9), and the methodology used to assess emotional changes range from clinical interviews and questionnaires (n. 13) to the recording of behavioural and autonomic responses (n.3) during experimental tasks (n. 6). 3 studies integrate self-referred responses and experimental tasks.

3.3.1. Emotional changes

Research into changes in the intensity of emotions after SCI are very few and inconsistent. While some studies report increased emotional reactions (Chwalisz et al., 1988; Davidoff et al., 1990), others document a reduction (Bermond et al., 1991). These differences might be due to the different typologies of responses considered, with increased scores related to cognition-mediated emotional responses on the one side and reduced reactivity linked to somatic responses on the other (Bermond et al., 1991; Buchtler et al., 2021).

Only one study has investigated various aspects relating to emotions in individuals after SCI (Saberi and Ghajarzadeh, 2017). The Emotional Quotient Inventory (Bar-On et al., 1997) was administered to a large sample of participants (110 people with SCI and 80 controls). The results revealed a degree of impairment in perception, understanding and the regulation of emotions in the SCI individuals. Crucially, a dissociation between emotions related to the self and social emotions emerged. Self-referred emotional experiences were found to be impaired, in particular, Intrapersonal states (Self-Regard, Emotional Self-Awareness, Assertiveness, Independence, and Self Actualisation), Stress Management (Stress Tolerance and Impulse Control) and Adaptability (Reality Testing, Flexibility and Problem Solving). Moreover, there was an association between the severity of the deficit and higher NLIs. In contrast, no differences between the SCI participants and the healthy controls emerged with regard to social abilities such as Interpersonal relationships, Empathy and Social Responsibility (Saberi and Ghajarzadeh, 2017).

3.3.2. Anxiety and depression

Anxiety, depression (e.g. Davidoff et al., 1990; Kennedy and Rogers, 2000; Saunders et al., 2012; Sher-Wei Lim et al., 2017) and post-traumatic stress disorders are commonly found in individuals with SCI (Hagen, 2015, see also Table 1). Although emotional adjustment processes may occur over time (Kennedy et al., 2000; Kennedy and Rogers, 2000; with longitudinal data of the same participants in Kennedy et al., 2016), depression is reported in around a quarter of people who have suffered SCI (22.1% in Saunders et al., 2012; 28.9% in Dryden et al., 2005) and is associated with a risk of alcohol or substance abuse (Williams et al., 2014) and mortality (Kennedy et al., 2016).

3.3.3. Emotion discrimination and learning

In terms of the identification of emotional stimuli, a dissociation between the recognition of facial expressions and the identification of emotionally evocative scenes is reported, with the patients with SCI being less accurate than the controls in the latter task (Pistoia et al., 2015). Furthermore, differences between SCI individuals and controls have been reported relating to scenes evoking fear or anger, but none have been found for other emotions (i.e. happiness, sadness, disgust and surprise). However, a similar task administered to patients with Locked-in syndrome (Pistoia et al., 2010) indicates the opposite pattern with deficits in this clinical population relating to facial expressions but not to the discrimination of scenes evoking emotions. It is clear that this issue needs further investigation.

Differences in emotion-related brain activity in SCI patients as compared to the controls were also found in an emotional learning paradigm during which the observation of stimuli showing angry faces was associated with the administration of an electrical shock to the upper arm. The results showed enhanced activity in the anterior cingulate and periaqueductal gyrus (reflecting the central sensitisation of the pain matrix) and decreased subgenual cingulate activity. The authors interpreted this latter finding as representing a possible substrate underlying the affective vulnerability present in SCI patients, consequent to perturbations in autonomic control and afferent visceral representations (Nicotra et al., 2006).

3.3.4. Bodily-autonomic functions in emotions

Direct investigations of the correlation between the perception of internal states of the body (i.e. interoception) and emotions are meagre. A first study (Montoya and Schandry, 1994) focused on heartbeat perception and found reduced ability in SCI participants associated with reduced self-reported emotional experiences. In addition, according to other studies, higher cardiac awareness correlates with more intense emotional experiences, in particular with regard to fear (Chwalisz et al., 1988) and sadness (Chwalisz et al., 1988).

Nevertheless, these results have been questioned in a more recent study that investigated responses to pictures evoking emotions (Cobos et al., 2002). No differences between the SCI participants and the controls were found in relation to the heart rate modulation (i.e. the unpleasant pictures produced greater deceleration than the pleasant ones), or in the valence and arousal ratings relative to the intensity of emotional experiences. Furthermore, no effects connected to the NLI, the interval from lesion onset or gender were found. However, skin conductance modulation induced by the nature of the stimuli (unpleasant pictures versus pleasant and neutral ones) was recorded in the controls but not in the SCI participants.

4. Discussion

Our review of the literature indicates that a number of cognitive and emotional changes occur as a consequence of body/brain disconnections following SCI. Previous studies on animal models have demonstrated the

role of the spinal cord in learning, in particular in motor learning (e.g. Grau et al., 2022), and experimental data on individuals with SCI have shown a reorganisation of those cognitive functions that are grounded on the information coming from the body (i.e. body, space and action representations, Moro et al., 2022). The current results expand on previous evidence which has shown that the cognitive changes occurring after SCI also involve cognitive functions which are apparently not directly associated with the body.

The idea that higher order functions such as executive functions or emotion discrimination can be impaired as a consequence of "peripheral" (i.e. out of the brain) damage is outside the dominant cognitivist vision of the human mind according to which the sensory and motor systems are entry and exit routes for information which is only centrally processed at a symbolic level (e.g. Newell and Simon, 1972; Leshinskaya and Caramazza, 2016). Instead, the results emerging from this scoping review of the literature encourage an embodied vision of knowledge, supporting the crucial contribution of bottom-up processes (from sensory-motor systems to cognition, from the body to the brain) to an individual's mental life and their interplay with cognitive processing.

Our analysis suggests a general framework which is in some ways common to cognition and emotions, indicating that pre-lesional, consolidated capacities resist the impact of the damage better than the fluid capacities. Indeed, fluid cognition (i.e. executive functions and working memory) is more compromised than crystallised cognition (i.e. long-term learning), and self-referred emotions (e.g. stress tolerance) are more impaired than learned, social emotions (e.g. social responsibility). When flexibility is required or when new responses need to be generated (but not when previously learned responses are available), SCI people find themselves in difficulty. From this perspective, the framework which emerged from the present study seems to support a point of view according to which body-brain connections are necessary for the individual's mental life. The imbalance between previously acquired knowledge and the moment-by-moment processing of new information is also consistent with data indicating that during cognitive and emotional tasks, individuals with SCI tend to rely on cognitive strategies rather than on the simulation of bodily states (Fiori et al., 2014; Guadagni et al., 2019) and show differences with respect to controls only when implicit measures are considered, but not when semantic, explicit responses are required (Scandola et al., 2020). For example, in a recent study on empathic responses to observing other people's pain (Scandola et al., 2023 in press), explicit responses (i.e. verbal judgments) and implicit responses (muscular – i.e. Corrugator muscle - and autonomic reactions - i.e. electrodermal response and Respiratory Sinus Arrhythmia) were recorded while participants were observing videos showing stimuli which were either painful or neutral administered to a hand, a foot or a control object. The results show that while explicit, verbal responses to pain do not differ in the two groups (the SCI participants and the control group), the SCI individuals' implicit responses are reduced.

4.1. Changes in cognition

Although this is, at least in part, unexpected, the independence of cognitive disorders from the occurrence of TBI is in line with the embodied approach to cognition. Conversely, contradictory results have been found with respect to the effects of the NLI on cognitive functions. Indeed, one might expect more severe disorders to be associated with higher lesions (i.e. in tetraplegia) and with Locked-in Syndrome than is the case with lower lesions (i.e. paraplegia). However, the lack of conclusive results and the ambiguous findings resulting from neuropsychological assessments has meant that it is not possible to confirm this hypothesis (Cohen et al., 1992, 1996; Allain et al., 1998; Sachdeva et al., 2018; Molina et al., 2018; 2021; Chiaravalloti et al., 2020). Motor deficits make this issue even more difficult to disentangle. Indeed, if on the one side it has been shown that even minimal motor deficits might impact performance (Lee et al., 2021), it is also clear, on the other side, that tasks involving multiple choice answers (e.g. indicating the correct response among a selection shown on a screen) are simpler than open questions or those for which no cues are provided (e.g. involving verbal responses). Response modalities during cognitive tasks represent a topic that deserves further investigation to reach a better understanding of the effects of lesion level irrespective of motor impairment. To date, to the best of our knowledge, no studies have directly compared motor and free-motor responses in the same participants.

It is worth noting, however, that most of the research reviewed in the present study documents the presence of deficits in psychometric measures (Table 1) and changes in brain activity when implicit measures are considered (Cohen et al., 1992, 1996; Vastano and Widerstrom-Noga, 2023) or measures such as electrophysiological activity during attentional tasks (Cohen et al., 1992, 1996) or mental imagery tasks (Vastano and Widerstrom-Noga, 2023) are taken into consideration. It is also important to underline that, in daily life, the majority of these patients maintain their autonomy.

This inconsistency between behavioural and psychometric or neurophysiological data may be due to the fact that individuals with SCI activate networks which are different to those activated in healthy subjects in order to execute cognitive tasks relating to daily life. The availability of these alternative networks represents a protective factor after lesions, in keeping with the notion of Cognitive Reserve (Stern, 2009). Cognitive Reserve is built during a person's lifespan and is influenced by education and social engagement, exactly the same factors that impact on cognitive decline in people with SCI when they are of a low standard (e.g. a lower standard of education, a history of smoking or abuse of substances or alcohol or sleep disorders) (Pasipanodya et al., 2021; Sachdeva et al., 2018). From this perspective, individuals with a greater cognitive reserve are better able to compensate for the effects of damage.

Neuroanatomical studies support this hypothesis (i.e. the use of alternative networks during cognitive tasks). They have shown that post-lesional plasticity extends beyond the sensory-motor spinal and cerebral networks (Freund et al., 2011; Kamble et al., 2011; Choe et al., 2017; Grabher et al., 2017) and involve associative cortices (Kokotilo et al., 2009; Solstrand Dahlberg et al., 2018) and functional connectivity (Zhu et al., 2015), with pain also playing a relevant role (Wrigley et al., 2009; Gustin et al., 2010, 2014; Jutzeler et al., 2015). In a study using whole brain diffusion tensor imaging (DTI) involving a relatively large group (32 chronic SCI individuals and 70 control subjects, Ilvesmäki et al., 2017), neurodegenerative processes were found not only in the projection tracts (i.e. thalamocortical projection, cerebral peduncle, internal capsule, and the white matter underneath the primary motor and sensory cortices), but also in the corpus callosum and the long tracts of association fibers (inferior and superior longitudinal fasciculus, inferior fronto-occipital fasciculus, uncinate fasciculus and anterior cingulum; Ilvesmäki et al., 2017). Unfortunately, no study has thus far correlated neuroanatomical changes with cognitive performance and, as a result, only indirect speculation is possible on the potential links between them.

It is also worth noting that, to date, studies investigating the effects of clinical variables on cognition (i.e. NLI, interval from lesion onset, drug use, pain, vital aspects such as cardiac function, respiratory aspects and gastric activities) are lacking. Likewise, studies addressing the effects of cognitive deficits on motor recovery (e.g. attention, Costa et al., 2016; Tse et al., 2017; Srisim et al., 2017; Amatachaya et al., 2019; visuo-spatial abilities, Scandola et al., 2016, 2020) are meagre despite the potential impact of cognitive aspects on recovery.

Taken as a whole, the results from research into cognitive changes following SCI indicate the need for further, in-depth investigations that take into account the extensive inter-individual variability that exists. They should also consider associated clinical factors and integrate behavioural, neurophysiological and neuroanatomical measures.

4.2. Changes in emotions

Seminal studies on potential changes in emotional experiences following SCI date back to the 60 s (e.g. Mueller, 1962; Hohmann, 1966) and report specific emotional reactions to the damage suffered (i.e. depression, despair, bitterness and grief; Mueller, 1962) as well as generally reduced arousal relating to emotional stimuli (i.e. fear, anger and sexual excitement). These changes were described as being more evident in individuals with higher NLIs (Hohmann, 1966; Jasnos and Hakmiller, 1975; Higgins, 1979). It should be noted, however, that later investigations have had results which are inconsistent with previous findings (e.g. Chwalisz et al., 1988; Bermond et al., 1991; O'Carroll et al., 2003; Nicotra et al., 2006; Deady et al., 2010; Buchtler et al., 2021). This discrepancy may depend on multiple factors, such as the domains investigated (e.g. anxiety/depression, emotion recognition and body related emotions), the methods used (e.g. retrospective studies and experimental or self-reported responses) or the characteristics of the samples recruited (i.e. in terms of NLI and completeness of lesion, and the interval since lesion onset, Table 2).

Taken as a whole, the results of this review reveal that experimental research on individuals with SCI has mostly neglected the domain of emotions. Only reactive emotional states such as depression and anxiety have been systematically investigated and are generally documented after lesion onset. One could argue that since in this condition the extent of functional damage is so great and the changes to the individual's daily life are so extensive, these aspects alone are enough to cause emotional reactions.

However, there are also data documenting changes in emotion discriminations (Cobos et al., 2002; Pistoia et al., 2010, 2015) and learning (Nicotra et al., 2006). Despite the clear necessity to further investigate these aspects and replicate the results, the findings of these studies seem to support the crucial role of the body in mental life. It is well known that in healthy people bodily states change as a consequence of emotional stimuli, and that these changes influence an individual's feelings and decisions (Damasio, 1996). Multisensory integration is altered in SCI patients who have lost the below-lesion somatic afferences (Vastano and Widerstrom-Noga, 2023) and the balance between sympathetic and parasympathetic responses regarding internal visceral states. Surprisingly, data on the perception of the internal states of the body in individuals with SCI are lacking, and little attention has been dedicated to the potential impact of interoception in emotional response modulation. The few studies on this topic found in the present review are inconclusive (Chwalisz et al., 1988; Montoya & Sandry, 1994; Cobos et al., 2002). Nevertheless, the deafferentation characterising these clinical conditions lends support to the hypothesis that patients will potentially be subject to subclinical emotional changes that, even if they are not detected by means of interviews and self-reporting, may in any case impact on individual's quality of life.

4.3. Conclusions and future directions

The review of literature presented in this study indicates that cognitive and emotional changes may occur following body/brain disconnections, thus underlining the importance of the role of body afferences and efferences in the individual's mental life. From a clinical perspective, this finding opens new scenarios in terms of diagnoses and interventions, in particular if the impact of subclinical cognitive changes on motor functions and autonomy is confirmed. Nevertheless, to date, the results have been insufficient and unclear, not only due to the fact that the evidence is inconsistent, but also because there is a lack of systematic controls of the numerous co-occurrent variables implicated in these processes. Further research should fill this gap with studies recording both subjective responses (i.e. questionnaires) and objective responses (i.e. experimental tasks and behavioural measures), in addition to body-related neurophysiological and neuroanatomical measures. This will make it possible to investigate cognitive and emotional changes after body-brain disconnection and explore various different levels of responses and potential compensative processes. Furthermore, social variables, such as social context and isolation, should be taken into consideration, in particular when addressing long-term changes in cognitive abilities. Crucially, if these results are confirmed, the implication is that there is an association between the risk of cognitive decline and lower performance during the acute phase. This means that it is vital to introduce cognitive assessments and emotional screening as part of the standard clinical practice during the post-lesional acute phase.

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Disclosure statement

The authors report there are no competing interests to declare.

Data Availability

Data sharing not applicable – no new data generated.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.neubiorev.2023.105395.

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